CAUSES OF BIGHORN SHEEP MORTALITY AND DIE OFFS
– LITERATURE REVIEW –

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Causes of Bighorn Sheep Mortality and Dieoffs

- Literature Review[1] -

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INTRODUCTION

The Rocky Mountain bighorn sheep (*Ovis canadensis canadensis* Shaw) populations of North America have markedly decreased in number and distribution since the human settlement of their native habitat (Buechner 1960). Factors responsible for the decline are suggested to include heavy hunting pressures, the introduction of domestic animal diseases and, especially in this century, the widespread reduction of available and suitable ranges (Buechner 1960; Stelfox 1971).

More recently, bighorn sheep have suffered epizootic dieoffs in captive and wild situations in Canada and the U.S.A. The sheep have nearly always succumbed to pneumonia caused by opportunistic bacteria with varying degrees of lungworm involvement (Forrester 1971). Reports of sheep dieoffs have outlined a large number of environmental and animal-related factors that have been present before dieoffs and were believed to predispose sheep to disease. For example, Spraker *et al.* (1984) described a series of conditions preceding a dieoff at Waterton Canyon in Colorado. These included increased levels of vehicular traffic, noise, human harassment, high animal density and a reduction in forage quality and quantity. Reports of other dieoffs have included factors such as low trace mineral levels in tissue, inclement weather, habitat deterioration and loss, the presence of domestic sheep, concurrent infectious diseases and high lungworm or other parasite levels (Buechner 1960; Lange *et al.* 1980; Foreyt and Jessup 1982; Thorne *et al.* 1982; Schwantje 1983; Onderka and Wishart 1984).

Such factors are believed to function through direct and indirect mechanisms, their presence and effect varying with each situation. Chronic exposure to some of these conditions may act cumulatively to impair protective immune functions. The collective influence is believed to create a state of chronic stress (Spraker *et al.* 1984). The physiological responses of individual animals to chronic stress include adrenal gland enlargement and increased levels of adrenal corticosteroids (Feldman 1983). Long term maintenance of high levels of corticosteroids in other species is associated with an inhibition of immune responses and increased susceptibility to bacterial infections, particularly of the respiratory tract (Hunningshake and Fauci 1977; Kelley 1980). Studies in man and domestic animals have shown that concurrent viral respiratory infections (Yates 1982), malnutrition (Scrimshaw *et al.* 1968) and low tissue levels of selenium and copper (Chandra 1983) are associated with, and can directly cause, depressed immune responses.

The proper function of immune systems is closely related to the overall nutrition of an animal (Chandra and Newberne 1977). Many of the factors discussed as influencing bighorn sheep herds play important roles in their nutritional condition. Forage quality and quantity have obvious direct

influences. Other factors, such as intra and interspecific competition, harassment and high gastrointestinal parasite burdens may indirectly reduce the amount and type of feed consumed and utilized. Indices of nutritional status have been examined in controlled studies (Kirkpatrick *et al.* 1975; Seal *et al.* 1978; Bahnak *et al.* 1979; Warren *et al.* 1982) and studies of free-ranging white-tailed deer (*Odocoileus virginianus*), pronghorn antelope (*Antilocapra americana*) and wild sheep (Franzmann 1972; White and Cooke 1974; Seal and Hoskinson 1978; Hebert *et al.* 1984; Spraker *et al.* 1984). The levels of total serum protein (TP), blood urea nitrogen (BUN), fecal nitrogen (FN) and body condition scores varied with season, habitat and physical condition in white-tailed deer and bighorns (Franzmann 1972; Seal *et al.* 1978; Hebert *et al.* 1984). These parameters were the most frequently used in these studies and were easily available from live animals.

Depressed TP has long been accepted as occurring with protein-energy malnutrition in man (Sauberlich 1983), and studies have demonstrated reductions in TP with starvation in wildlife species (Hebert 1973; Bahnak *et al.* 1979; Warren *et al.* 1982). Blood urea nitrogen levels were proportional to protein consumption in cattle (Biddle and Evans 1973), domestic sheep (Preston *et al.* 1965), white-tailed deer (Seal *et al.* 1972; Kirkpatrick *et al.* 1975) and pronghorn antelope (Seal and Hoskinson 1978). Franzmann (1972) stated that the nutritional status of bighorn sheep was best estimated by BUN and suggested that values below 15 mmol/L indicated low protein intakes.

Comparisons of fecal nitrogen content with feed nitrogen content have been made to assess the protein status of wildlife populations. Species examined include free-ranging elk (*Cervus elaphus*) (Gates and Hudson 1979), East African ungulates (Arman *et al.* 1975) and wild and captive bighorn sheep (Hebert 1973; Hebert *et al.* 1984). In most cases, studies have followed seasonal trends in FN and related these to environmental changes (Seip and Bunnell 1985), feed quality and animal weight (Mould and Robbins 1981). Hebert *et al.* (1984) showed changes in animal condition between seasons and years in captive, supplementary fed, free-ranging and non-migratory free-ranging California bighorn sheep (*O. c. californiana*). They advised using FN determination over time to assess the effect of changes in population density and grazing on sheep ranges and herds. Bahnak *et al.* (1979) suggested that periodic monitoring of all nutritional indices was more valuable than conclusions drawn from samples taken at a single point in time.

The definition of trace mineral deficiencies requires the association of clinical signs of deficiency with low mineral concentrations in animal tissues, diet and soil. Deficiency syndromes are often subclinical and may be difficult to identify due to interactions and imbalances between individual minerals and the protein-energy content of the diet. Studies related to two of the more commonly recognized deficiencies, selenium and copper, are limited in free-ranging wild species; however, both minerals have

been extensively reviewed for domestic cattle and sheep (Hidiroglou 1979; Underwood 1977; Van Vleet 1980).

Subtle or marked syndromes of muscle degeneration, ill thrift, neonatal weakness and infertility are responsive to the addition of selenium and vitamin E (Se/E) to the diets of ruminants (MacDonald *et al.* 1976; Maas 1983). In addition, suppressed cell-mediated immune functions are restored (Sheffy and Schultz 1979). An acute stress-induced muscle degeneration (capture myopathy) has been well recognized in wild ungulates and is associated with Se/E deficiency in some instances (Hebert and Cowan 1971). The other livestock syndromes are not well defined in wild species but are suspected to occur.

Copper deficiency in domestic sheep is also often subclinical but can reduce growth rate, reproductive performance and haircoat pigmentation (Ward 1978; Hidiroglou 1979). Studies in Alaskan moose (*Alces alces*) and Idaho mule deer (*Odocoileus hemionus*) suggested that these were reductions in fertility and abnormal hoof keratinization (Flynn *et al.* 1977; Dunbar and Foreyt 1985). Low copper levels are also associated with decreased immune responses and an increased susceptibility to parasitism (Chandra 1983). A possibility of interaction between copper and selenium has been proposed because of a lack of response to supplementation until both elements were provided to domestic livestock (Blood *et al.* 1983).

In this report, I review the history and causes of disease and mortality in the wild sheep of North America, outline the principle of synergism of infectious agents and environmental factors in domestic animal pneumonia and associate it with a similar pathogenesis of the respiratory disease complex of Rocky Mountain bighorn sheep.

This project was conceived, designed and carried out as a cooperative investigation involving numerous individuals and groups from the private, academic and government sectors, all sharing a special interest in that remarkable animal, the bighorn sheep.

Particular appreciation is expressed to the Foundation for North American Wild Sheep, the British Columbia Guide-Outfitters Association and the B.C. Ministry of Environment and Parks, Wildlife Branch for their financial support. The B.C. and Alberta Ministries of Agriculture, the Canadian Animal Disease Research Institute, W.M. Samuel, T.R. Spraker and A. Gajadhar generously donated analytical services. Invaluable field assistance and advice was provided by P.W. Davidson, R. Demarchi, T.R. Spraker, A. Wolterson-Strauss, C. Shepstone, D. Hebert and the hunters of the East Kootenays. Crowsnest Resources and G. Schuerholz permitted access to sheep range and their study data.

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BIGHORN SHEEP DIEOFFS

NORTH AMERICA

The most widely distributed wild sheep species in North America is the Rocky Mountain bighorn sheep. Estimates of bighorn distribution and numbers prior to the settlement of western North America are few, but sheep were reported to be present over wide areas of what is now western Canada and the United States. Counts of up to 2 million bighorn sheep for the U.S.A., Canada and Mexico prior to 1800 have been suggested (Buechner 1960). This absolute figure is probably of little value due to inaccurate census methods, but does serve to contrast dramatically with counts of less than 20 000 bighorns in the U.S.A. in 1960 (Buechner 1960).

Bighorn population declines occurred as human-related activity was introduced into accessible bighorn habitat. Buechner (1960) attributes the majority of sheep losses in the last half of the nineteenth century to several factors acting singly or in combination. These included excessive hunting with firearms, disease epizootics, competition from livestock and restriction of winter range.

Legislation to preserve sheep habitat, reduce native predators and to prevent hunting was passed by wildlife agencies in the late 1800's and early 1900's. These actions, as well as reintroductions of sheep into empty U.S. ranges in the 1950's and attempts at habitat restoration, were successful in slowing declines and increasing numbers in some areas. Many populations, however, continued to decline and remained as small remnant herds or vanished. Although specific causes were never determined, the disappearance of some herds coincided with the intensified use of bighorn habitat for livestock grazing and recreational, urban and industrial development. It is believed that another major factor in the loss of sheep habitat was the effective prevention and control of fires (Demarchi 1972). Grassland ranges previously maintained by periodic natural fires underwent successional growth by conifer trees or were allowed to overgrow with non-forage plant species. These processes combined to reduce available range.

Certain diseases were noted in the sheep population as a whole and were believed to be responsible for at least some of the initial losses of bighorn sheep. Reports of bighorn mortality occurring in large scale epizootics were referred to as dieoffs. The earliest dieoffs were attributed to scabies mange caused by *Psoroptes ovis*. This external parasite was considered to have been brought to bighorn ranges by domestic sheep herds (Buechner 1960). Later dieoffs of bighorns in the U.S.A. were reported to have a common disease pattern, almost exclusively of fatal acute and chronic pneumonia, caused by opportunistic bacteria such as *Pasteurella* spp.. A lungworm, *Protostrongylus* spp., unique to mountain sheep, was involved in most

cases. This bacteria-lungworm syndrome became known as the lungworm-pneumonia complex of bighorn sheep (Forrester 1971), but is perhaps more accurately referred to as the respiratory disease complex of bighorn sheep (Onderka and Wishart 1984).

The serious influences of disease dieoffs on bighorn herd production have stimulated much interest in the species. Research, particularly in the past 10-20 years, has associated a wide range of animal and environmental conditions with outbreaks of pneumonia. The combination and overall importance of these factors has varied with each herd situation.

General types of dieoffs have been described. In addition to the classic "all-age" dieoffs with mortality initially seen in juvenile and adult sheep in good body condition, there are lamb dieoffs classified as summer lamb pneumonia and stress-induced lamb pneumonia (Spraker 1979). Other, primarily adult dieoffs, have been described on the basis of the prominence of specific factors such as severe lungworm infections (Spraker 1977), varying degrees of malnutrition, human harassment (Spraker *et al.* 1984), interspecific competition and adverse weather conditions. These dieoff classifications will be discussed in detail.

CANADA/EAST KOOTENAYS

Rocky Mountain bighorn sheep in Canada are concentrated in the Rocky Mountain ranges on both sides of the British Columbia/Alberta border. The development of this area for human use has largely been restricted in comparison to the mountainous areas of the U.S.A.. In fact, much of the area occupied by bighorns is now protected under National and Provincial park status. Bighorns within park boundaries are not hunted and their ranges are protected from most human-related encroachment, as well as from natural environmental changes such as fires. Sheep living outside of the park systems for most of the time are currently managed in accordance with provincial big game hunting regulations. Provincial management practices have focussed on range protection and improvement, mostly by manipulation of critical fall and winter ranges.

The human-related pressures responsible for declines in bighorn numbers in the U.S.A. have also caused reductions in overall numbers of Canadian bighorns. The population declined from 10 000 on the eastern slope of the Rockies in 1850, to 2600 in 1915 (Bandy 1968; Stelfox 1971). Stelfox (1971) reviewed the dynamics of Rocky Mountain bighorns in this area and demonstrated a markedly fluctuating pattern. During the period 1915-1936, the depleted population increased to 8500. The area had been completely closed to hunting and this was strongly enforced. Livestock grazing was eliminated or reduced from mountain grasslands. Other game, such as bison (***Bison bison***) and elk were not particularly abundant and it is thought

that extensive fires in the late 1800's and early 1900's expanded grasslands on bighorn ranges.

All major park bighorn populations increased but these increases were followed by rapid declines. In some instances, 75% or more of the population died, with many of the declines shown to be due to lungworm-related pneumonia. By 1950, the total population of bighorn sheep had again reached 2500 (Stelfox 1971). Although no sequential pattern was noted, Stelfox (1971) outlined similar high densities, heavy interspecific forage competition resulting in range deterioration, severe winter weather and heavy lungworm levels prior to dieoffs in affected herds. With mild weather conditions and the reduction of grazing pressure on ranges after the dieoffs, sheep numbers increased once again to 10 000 by 1970 (Stelfox 1971).

The East Kootenay (E.K.) region of British Columbia contains 85% of the Rocky Mountain bighorn sheep in the province, and consists of from 1500 to 2000 animals. The population can be subdivided on the basis of winter range location into at least 20 herds. Twelve of these herds winter on low elevation, southwestern-facing slopes on or near the eastern side of the Rocky Mountain Trench. An additional eight or more herds winter at high elevations surrounding the upper Elk River valley (Figure 1).

The sheep in the E.K. have experienced large scale all-age dieoffs in the 1920's, 1940's, 1960's and 1980's. Although detailed records are not available for each dieoff, local interest preserved some facts. Certain common circumstances have been associated with the mortality.

The herds reported to have undergone dieoffs were those that were relatively accessible due to their habitual use of low elevation winter ranges in or adjacent to the Rocky Mountain Trench. Little information has been available on the status and mortality patterns of the herds wintering at high elevation. The inaccessibility of many of the ranges has prevented detailed observations; however, the area has been utilized frequently for hunting and has been extensively explored by mining interests. None of these sources have reported sick or dead bighorn sheep in large numbers. More intensive aerial surveys and biological studies in the area since 1971 have shown no radical changes in the high elevation herds (W. Warkentin, pers. comm.; Schuerholz 1984).

The affected herds usually had population growth patterns that reached a population density considered to be high by biologists working in the area just prior to disease outbreaks. There was an interval of 16 to 25 years between dieoffs. In almost all cases, the animals that were found dead or seen ill were of mixed age and sex. Those seen ill had obvious clinical signs of respiratory disease. The initial deaths in each outbreak were peracute and usually occurred during the fall or winter, although animals with chronic pneumonia were found in later stages of the dieoffs. Postmortem

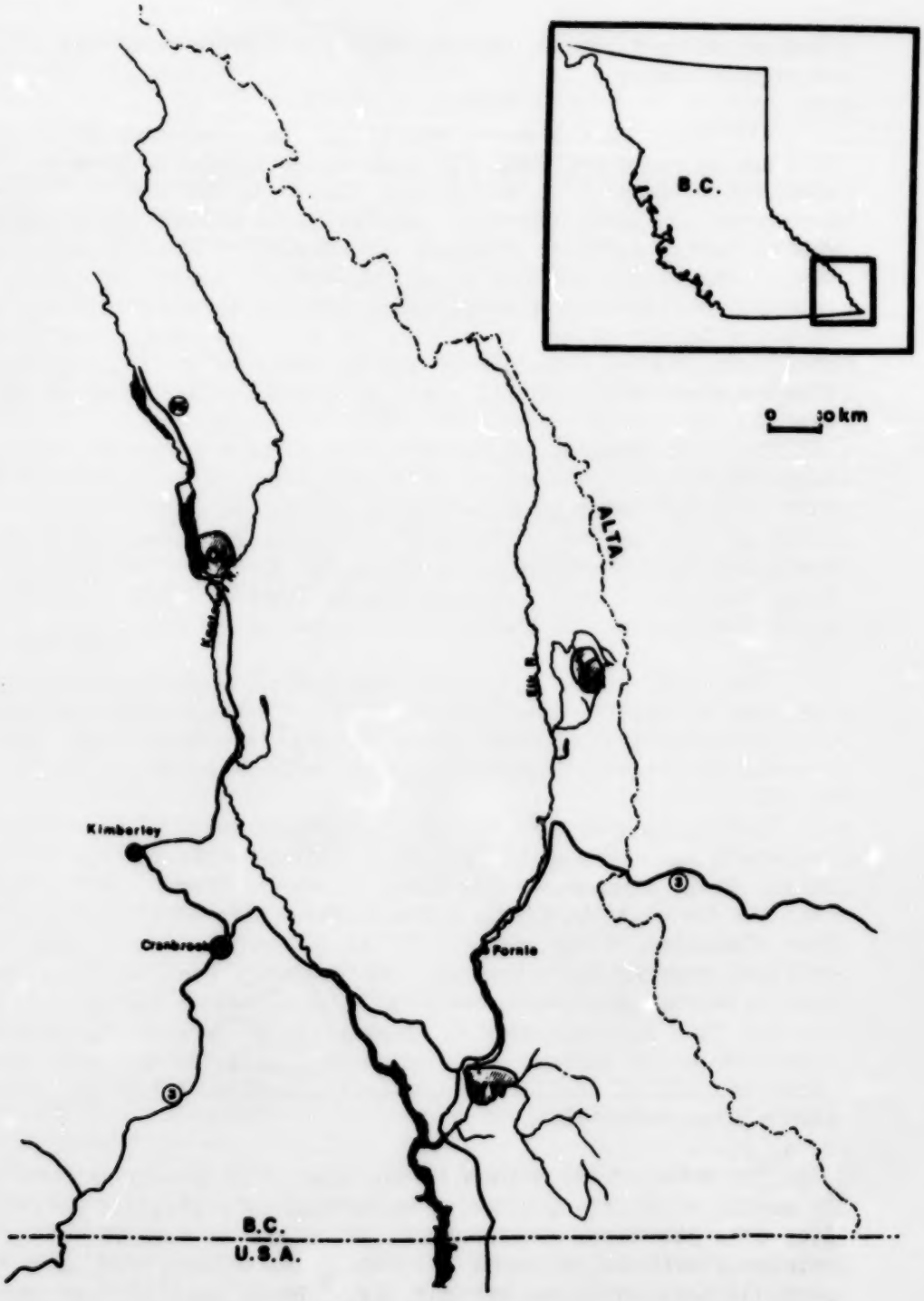

Fig. 1. Study area in southeastern British Columbia.

examinations performed on some of these sheep (Bandy 1966; Davidson 1982) confirmed diagnoses of bacterial pneumonias complicated by mild to severe lungworm infections. Gastrointestinal parasites and malnutrition were also present in various degrees. In addition, the dieoffs followed contact of bighorn sheep with domestic sheep in 1942, 1965 and 1981. No further investigations were attempted to investigate the presence of respiratory diseases in the domestic sheep herds. Heavy snowfalls and wet, early summers, which provided excellent conditions for snails (intermediate hosts of *Protostrongylus* spp.), preceded dieoffs in 1964 and 1981. Finally, the dieoffs in the E.K. were rarely confined to one herd but tended to occur sequentially through many of the herds. With better census techniques and observations during the 1960's and 1980's, the multiple herd nature of dieoffs in the E.K. became more obvious.

The first bighorn dieoff in the E.K. to be investigated intensively began in December 1964 at the Bull River winter range in the Rocky Mountain Trench. Strong winds, a heavy snowfall with a thick crust and poor winter range as a result of heavy big game and livestock grazing pressure limited feed intake and quality. The sheep were allowed free access to alfalfa hay on a neighbouring farm. Rumen impaction with the unnatural feed resulted. During the winter, over 95% of the herd died of acute or chronic bacterial pneumonia complicated by heavy lungworm infections (Bandy 1966; Demarchi 1972). Similar respiratory disease reduced the Wigwam herd by 38-50%, Premier Ridge herd by 72%, Columbia Lake herd by 73% and Stoddart Creek herd by 76% over the next 3 years (Bandy 1968). Other adjacent herds were also affected. The total number of bighorns in the E.K. dropped to 720 from a high of at least 1650 in the early 1960's (Demarchi 1972).

Recovery efforts over the next decade and a half were concentrated on aggressive habitat management, acquisition and protection programs, as well as the reduction of elk numbers in strategic areas such as the Wigwam winter range. Sheep numbers responded, climbing to pre-1964 numbers in most areas.

Previous sheep and other big game density and disease patterns suggested the development of a dieoff in the Wigwam range in the 1980's. The situation was expected to be critical by 1985, 20 years after the last dieoff. However, the bighorn sheep in this herd increased more rapidly than expected, reaching approximately 424 (P. Davidson, pers. comm.) by the spring of 1981. Maximum counts of only 174 (Bandy 1968) preceded the 1960's dieoff. During the late fall of 1980, lambs on the Wigwam range were observed with oral lesions of contagious ecthyma. Two-thirds of the lambs subsequently disappeared over the winter months. In the second week of December 1980, animals in good condition began dying acutely on the adjacent Maguire Creek range. The Maguire Creek bighorn sheep had shared this good quality range with domestic sheep during the fall of 1980. All of the carcasses examined had lesions of bacterial pneumonia with prominent lungworm infections.

Sixteen sheep from the Wigwam range were captured live, given prophylactic antibiotics and a vitamin complex and released at Bull River in March 1982. Eight of these sheep died within 6 weeks with respiratory disease similar to subsequent deaths on the Wigwam range. None of the resident Bull River herd became ill in spite of thoroughly mixing with the transplants.

In the following 2 months, at least 46% of the Wigwam herd died with acute to chronic pneumonia, many having light lungworm infections (Davidson 1982). Serum and liver selenium and copper levels were interpreted to indicate deficiencies of these elements in some sheep. Over 80% of 85 carcasses that were aged and sexed were between 4 and 7 years of age. A large proportion of the losses in this herd then consisted of the prime reproductive age class. Chronically affected sheep were occasionally present on the range in the following years. Lamb production in the surviving sheep has been extremely poor with small size and high mortality observed in lambs less than 6 months of age.

Over the next 2 years, there were reports of thin and/or coughing sheep at Premier Ridge, Wildhorse and Lussier ranges. In the opinion of the local biologists, the number of bighorn sheep declined in these and small associated herds. Several carcasses examined had "classic" lesions of the bighorn respiratory disease complex (R. Lewis, pers. comm.).

The occurrence of disease in bighorn sheep in the E.K. has twice shown a progressive northward movement in the Rocky Mountain Trench wintering herds. In 1982-83 similar mortality was also seen in southwestern Alberta herds (Onderka and Wishart 1984). It can be suggested that contact between individuals of different herds can occur on common summer ranges or when wandering ram bands mix prior to or during the rut. Festa-Bianchet (1986) demonstrated that different ewe groups can share the same ranges at different times of year. This may result in another source of increased contact between groups of bighorn sheep. Mortality seen in adult sheep in the early fall may support the concept of transmission of types or numbers of pathogens between herds in summer months. It seems most likely that the pattern seen in the E.K. has a direct relationship to the large number of contiguous herds and the transmission of large numbers of pathogenic organisms between herds of differing susceptibility.

DIEOFF TYPES

As previously discussed, the mortality of free-ranging and captive bighorn sheep has demonstrated a remarkable epizootic pattern. Dieoffs have been associated with *Psoroptes* mange infections (Buechner 1960; Lange *et al.* 1980) and pneumonia (Marsh 1938; Spraker 1977; Spraker *et al.* 1984; Onderka and Wishart 1984).

Spraker and Hibler (1982) discussed the respiratory diseases of bighorn sheep and classified pneumonia mortality into three clinical types. The types are subdivided primarily on the basis of age with secondary criteria of seasonal occurrence, pathological characteristics and predisposing conditions.

The first type of pneumonia dieoff has been recognized since the 1800's and is commonly known as the "all-age dieoff". In this form, sheep of any age may be affected. The outbreak may occur in any season, but usually begins in fall on winter range and is associated with multiple environmental and density-linked stress factors, including various degrees of lungworm infection (Spraker *et al.* 1984). Many animals die acutely in good physical condition with fibrinous bronchopneumonia, but emaciated sheep with chronic lesions may die subsequently after protracted illness. All-age dieoffs in captive bighorns usually follow a similar pattern.

The organisms recovered from pneumonic lesions in this type of dieoff are generally considered to be opportunistic invaders. It is felt that a series of environmental and physiological events, which may vary among dieoffs, cause a state of chronic stress in the herd. This type of chronic stress causes adrenal cortical hypertrophy and hypersecretion of adrenal corticosteroids (Feldman 1983). Immunological responses are reduced, resulting in an increased susceptibility to pathogenic organisms (Harlow 1983). Factors such as increased human activity, vehicular traffic, noise, dust, reduced forage quality and quantity, inclement weather, high inter- and intraspecific competition and high levels of parasitism have been suggested as contributing to all-age dieoffs (Buechner 1960; Bandy 1966; Schwantje 1983; Onderka and Wishart 1984; Spraker *et al.* 1984).

Other types of dieoffs have been recognized as census and monitoring techniques have improved and have allowed the evaluation of the production of bighorn lambs and their survival to yearling status in some herds. Woodard *et al.* (1974) initiated a study in 1969 to investigate the declining Sangre de Cristo herd in Colorado. Over 2 years, they concentrated on the ewe-lamb segment of the herd and found that although ewe-lamb ratios were high in early July (72-83%), they were reduced to 17-22% by mid-September. No adult mortality was seen. The authors observed progressive symptoms of respiratory disease in lambs throughout the late summer in both years. The lambs were lethargic, weak, and had paroxysmal coughing and dyspnea. Necropsies performed on two lambs with these clinical signs showed large numbers of first stage *Protostrongylus* spp. larvae and secondary bronchopneumonia. Bacteria of several species, including *Pasteurella* spp. were isolated. The mortality was also associated with periods of cold, wet weather (Woodard *et al.* 1974). Descriptions of sick lambs during the summer have also been reported by Marsh (1938), Honess (1942) and Buechner (1960) in numerous locations. Spraker (1977, 1979) separated the syndrome into two types based on the presence of heavy lungworm infections.

The first of these has been termed "summer lamb pneumonia" and appears to occur following transplacental transmission of large numbers of *P. stilesi* from the ewe to the lamb *in utero* (Hibler *et al.* 1974; Spraker 1979). The parasite matures in the lungs and produces larvae by the time the lambs are 1 month old. Lung tissues damaged by the first stage larvae are secondarily invaded by an assortment of opportunistic pathogens. Acute pneumonia is believed to be precipitated by additional stressors, such as inclement weather, but chronic pneumonia is common. Hence the affected lambs may survive for variable lengths of time.

The second form of pneumonia seen in lambs is characterized by little to no lungworm involvement and has been classified as "stress-induced lamb pneumonia" (Spraker 1979). All of the herds involved suffered all-age dieoffs prior to the dieoff of lambs. Affected lambs born after the dieoffs had lesions of bacterial bronchopneumonia and the thymus was markedly reduced in size. Chronic stress is believed to be responsible for reducing the immune system functions of these lambs at an early age. Spraker *et al.* (1984) suggested that the stresses that precipitated the all-age dieoff in the adult sheep may affect the pregnant ewe and inhibit the development of the fetal thymus. In addition, the production of colostral antibodies may be diminished. Lambs would then be poorly prepared to encounter even normal microflora. Ewes suffering from chronic pneumonia may shed large numbers of bacteria that could overwhelm the immature defenses of lambs born with abnormal or normal thymic function.

BIGHORN MORTALITY - CAUSES

As previously discussed, the clinical disease and mortality of bighorn sheep have been most frequently caused by respiratory diseases (Buechner 1960; Forrester 1971; Spraker 1979). Fatal pneumonias usually include gross and microbiological evidence of bacterial and lungworm etiologies, but viruses and other organisms, such as mycoplasma and chlamydia, have also been recovered in some disease outbreaks (Pearson and England 1979; Woolf *et al.* 1970). Because bighorn sheep are susceptible to many diseases of domestic livestock, domestic animal pathogens are among those agents implicated in the decline of bighorn sheep populations (Foreyt and Jessup 1982; Goodson 1982). Dieoffs have frequently been associated with contact between these species and there apparently have been transfers of infection following interspecific association.

VIRAL

In recent years, increased awareness of synergism between infectious organisms in diseases of domestic species has given impetus to the search for multiple agents that might predispose bighorns to respiratory disease complex outbreaks. Serological surveys have indicated the exposure of many

North American populations of wild sheep to various respiratory and systemic viral diseases of wildlife and domestic animals.

Antibody titres to the respiratory viruses, parainfluenza virus type 3 (PI3), infectious bovine rhinotracheitis (IBR), bovine respiratory syncytial virus (RSV), as well as to bovine parvovirus 1 and adenoviruses have been present in all North American wild sheep species examined. Titres to PI3 were considered by the authors to be significant in Wyoming and Montana Rocky Mountain bighorns (Howe *et al.* 1966). Antibodies to PI3, bovine parvovirus 1 and adenovirus were present in a large proportion of bighorn samples from Colorado and Wyoming (Parks and England 1974). Desert sheep (*O. c. nelsoni*) herds in California frequently had high numbers of animals seropositive for PI3 (Clark *et al.* 1985), whereas only a few positive animals have been found among Dall sheep (*O. dalli dalli*) (Zarnke 1983).

Parainfluenza 3 virus has been isolated from both healthy and clinically ill free-ranging and captive sheep herds. Parks *et al.* (1972) reported an outbreak with mortality involving secondary bacterial pneumonia in Rocky Mountain bighorn sheep recently placed in captivity. The pattern of disease in these sheep suggested that previously exposed animals kept in close confinement infected others that lacked previous experience with the virus. Parainfluenza 3 virus was also isolated from free-ranging desert bighorn lambs with and without pneumonia (Jessup 1985). This virus is believed to be a contributing factor in the mortality of desert bighorn lambs in California (Deforge *et al.* 1982).

Parainfluenza 3 virus alone can cause acute pulmonary infections, but the uncomplicated infection in cattle and sheep rarely causes mortality (Dungworth 1985). Experimental studies have shown that the degree of lung damage is proportional to the virulence and dose of the virus and the resistance of the animal (Yates *et al.* 1983a). It appears that the most important role of the virus is to reduce pulmonary defenses, allowing the invasion of bacteria and the development of bronchopneumonia.

Little definitive information is available on bovine parvoviruses except that they are ubiquitous in cattle and appear to be most common in multifactorial diarrhea outbreaks in young calves (Barker and Van Dreumel 1985). Adenoviral infections are primarily associated with ocular or respiratory diseases and enteritis. Exposure to the virus is widespread in cattle populations but clinical disease is sporadic except in immunocompromised individuals. The significance of titres to either virus in bighorn sheep is unknown.

Clark *et al.* (1985) found widespread evidence of exposure of bighorn sheep to RSV throughout American sheep ranges. This virus was isolated concurrently with *Pasteurella haemolytica* type T from a single lamb with pneumonia in Colorado (Spraker *et al.* 1986). The significance of this

disease in bighorns is presently unknown but, with the identification of this case, it seems very likely that it may play a role similar to that in domestic cattle and sheep. There is evidence of its involvement in the bovine shipping fever complex, but it is also suspected to cause outbreaks and mortality as a single infection, perhaps more often than is the case with pure PI3 infections (Trigo *et al.* 1984; Lehmkuhl and Cutlip 1979).

Other ruminant viruses can also cause systemic disease with symptoms of respiratory infection. Surveys for antibodies against some of these, such as IBR, bovine virus diarrhea (BVD), bluetongue (BT), contagious ecthyma (CE) and epizootic hemorrhagic disease (EHD) have suggested occasional exposure of bighorn sheep (Dunbar *et al.* 1985).

Bluetongue and EHD are arthropod-borne viral diseases of warm temperate areas that affect domestic and wild ruminants. Bluetongue has a wide range of native wild hosts. Clinical disease is most severe in sheep and wildlife species. Inapparent infections do occur and are particularly frequent in cattle. Epizootic hemorrhagic disease has been most commonly seen in white-tailed deer (Kistner *et al.* 1975).

Bluetongue virus infection was demonstrated in a young wild desert ram with clinical respiratory symptoms (Robinson *et al.* 1967). Kistner *et al.* (1975) reported that the virus was isolated from an epizootic in penned California bighorn sheep and suggested that the virus originated from a nearby herd of domestic sheep. Desert bighorn sheep in Texas were greatly reduced in number during a period when a serious epizootic of BT occurred in domestic sheep (Robinson *et al.* 1967; Hailey *et al.* 1972). In California, captive and wild desert bighorn lambs with pneumonic disease consistently yielded several types of BT virus. In one case EHD virus was also isolated. Insect vectors for both BT and EHD are particularly common during lambing season (Deforge *et al.* 1986) in the specific area of California where the mortality occurred.

Contagious ecthyma is a viral disease of domestic sheep and goats and has been reported to occur as natural and experimental disease in various wildlife species, including wild sheep (Blood 1971; Samuel *et al.* 1975; Lance *et al.* 1981; Lance *et al.* 1983). It is characterized by proliferative lesions of mucous membranes and skin, in particular in and surrounding the oral cavity. The disease is usually more severe in young animals, primarily affecting them by interfering with suckling and prehension. Older animals lacking previous exposure to the disease, or under stressful conditions, have also been severely affected (Dieterich 1981).

Outbreaks of CE have been seen in captive (Dieterich 1981) and free-ranging wild sheep (Blood 1971; Samuel *et al.* 1975), and have occurred in sequential years in the same herd. Researchers have suggested that this pattern was due to "latency" properties of the virus (Hebert *et al.* 1977;

Lance *et al.* 1981; Zarnke and Dieterich 1985). Exogenous dexamethasone administration has resulted in reoccurrence of lesions in domestic sheep (Lance 1980), although the virus was not recovered from the resulting lesions. A similar attempt to reactivate the virus in two Dall ewes was unsuccessful (Zarnke and Dieterich 1985). Generally it is believed that cell-mediated immunity is involved in resistance to this disease (Zarnke 1984), and that compromise of this system may allow reactivation or more fulminating forms of the disease.

Serological evidence of exposure to CE was present in 9 of 18 herds of desert bighorn sheep (Clark *et al.* 1985) and 23-37% of Dall sheep (Foreyt *et al.* 1983; Zarnke *et al.* 1983). The disease has been reported repeatedly in Canadian Rocky Mountain bighorn sheep in both park and nonpark populations (Connell 1954; Blood 1971; Samuel *et al.* 1975; Davidson 1982). Persistent herd infections were associated with prolonged access to salt sources (Blood 1971; Samuel *et al.* 1975). High intraspecific competition, close contact of animals and the presence of an abrading substrate, such as salt blocks are believed to facilitate viral transmission (Lance *et al.* 1981).

BACTERIAL

The bacteria recovered from bighorn sheep include species causing specific diseases in both domestic and wild animals. Some of these bacteria are responsible for extensive bighorn mortality and have been associated with livestock contact (Goodson 1982). The majority of bacteria, however, cause only sporadic, usually localized infections. Although these may or may not be life-threatening, they are usually restricted to individuals rather than to groups of animals and are therefore of less importance to the management of wild sheep and not detailed here.

Pneumonia is the single most important disease condition of bighorn sheep. Bacterial agents isolated from pneumonic lungs of bighorn sheep include *Pasteurella haemolytica, P. multocida* (Foreyt and Jessup 1982; Marsh 1938; Spraker and Hibler 1977), *Bordetella* sp. (Thorne *et al.* 1982), *Neisseria* sp., *Hemophilus ovis, Actinomyces pyogenes* (Buechner 1960; Marsh 1938; Spraker 1979), *Streptococcus* sp., *Staphylococcus* sp. and numerous others (Spraker and Hibler 1982).

Of these, the *Pasteurella* spp. and *Actinomyces pyogenes* have been cultured most often, both from outbreaks that occurred early in the century, as well as from more recent pneumonia outbreaks (Buechner 1960; Spraker *et al.* 1984). The remaining species mentioned have been isolated with less frequency in various locales without any significant patterns being evident. Some species (*Streptococcus, Staphylococcus, Bordetella*) are considered to be of most significance as secondary invaders (Woolf and Kradel 1973; Thorne *et al.* 1982), but have also been isolated as sole agents in pneumonic lesions (Spraker 1979).

Most of these bacteria have been cultured from the upper respiratory tracts of healthy ruminants and are considered to be part of the normal microflora. They are of low virulence alone and require either predisposing damage to upper and/or lower respiratory tracts, or a reduction in normal immunity, to allow their multiplication and invasion of pulmonary tissues.

Pasteurella haemolytica, in particular, has been recognized to play a major role in the mortality of bighorn sheep, at least since Potts (1937) diagnosed "haemorrhagic septicemia" among bighorn sheep in Colorado. Marsh (1938) surveyed pneumonia dieoffs of Montana bighorn sheep from 1927-1935 and reported them to be due to verminous pneumonia. He also cultured both *Pasteurella* spp. and *Actinomyces pyogenes* from these lungs. Pneumonia outbreaks among captive and wild bighorn sheep occurring since this time in both the U.S.A. and Canada have consistently demonstrated lesions associated with the same bacteria, or yielded them on culture (Bandy 1968; Forrester 1971; Foreyt and Jessup 1982; Spraker *et al.* 1984). In fact, the reduction of wild bighorn numbers this century must be attributed in part to the apparently extreme sensitivity of this animal to *P. haemolytica*.

Pasteurella spp. bacteria are comprised of several species, each with strains differentiated on the basis of biochemical properties (Biberstein *et al.* 1960). Strains may vary in their prevalence and effects in different animal species. For example, *P. haemolytica* biotype A is usually isolated during bovine pneumonia, domestic sheep pneumonia and preweanling lamb septicemia; whereas biotype T causes septicemia in postweanling lambs (Dungworth 1985).

Two overall syndromes in domestic animals are caused by *Pasteurella* infections, haemorrhagic septicemia and pneumonia. Haemorrhagic septicemia is caused by *P. multocida* and has been reported in deer, cattle, swine, horses, bison and camels (*Camelus* sp.) (Dungworth 1985). The condition in domestic sheep is more often caused by *P. haemolytica* and is characterized by sudden death with vague clinical signs (Blood *et al.* 1983).

The pneumonic form is common in cattle. Although it may be seen with various management conditions, it is often associated with transport stress, hence the disease is often referred to as shipping fever (Blood *et al.* 1983). *Pasteurella haemolytica* is the bacterium isolated most frequently from pneumonic pasteurellosis of both cattle and domestic sheep; however, *P. multocida* may be present alone or in combination with *P. haemolytica*. Acute and chronic environmental stresses and viral respiratory infections have been described and experimentally induced to initiate the development of acute pneumonic pasteurellosis in these species (Biberstein *et al.* 1971; Cutlip and Lehmkuhl 1983; Friend *et al.* 1977; Yates 1982).

Both *Pasteurella* spp. can be recovered from the nasopharynx and tonsils of healthy bighorn sheep (Thorne *et al.* 1979; Marshall *et al.* 1983; Onderka and Wishart 1984). An atypical *P. haemolytica* biotype T has been especially common in pneumonic lungs from dieoffs in southeastern British Columbia and southwestern Alberta. The same biotype was also found in normal bighorn tonsils (Onderka and Wishart 1984; Spraker *et al.* 1986), and has been recently cultured from pneumonia cases in Colorado (Spraker *et al.* 1986). Isolates of biotype T from sick bighorn sheep and healthy domestic sheep tonsils produced fibrinous bronchopneumonia in captive bighorn sheep when inoculated intra-tracheally (Onderka 1986). Onderka (1986) also found that commercial intradermal biotype A bacterin caused septicemia and pneumonia in captive bighorn sheep within 3 days. It is not possible to compare the biotypes of recent and previous dieoffs retrospectively, since typing of *P. haemolytica* isolates was not done in the past. Both biotypes, however, appear to be capable of reproducing pasteurellosis in bighorn sheep under experimental conditions in the absence of other agents (Onderka 1986).

The development of pneumonic pasteurellosis in the bighorn sheep appears to follow the same general pattern as it occurs in domestic ruminants. Bacteria, normally resident within the pharyngeal region, multiply and subsequently invade the nasopharyngeal mucosa and lower respiratory tract. This change in the role of *Pasteurella* from commensal organism to pathogen is believed to be initiated by modifications in respiratory tract immune mechanisms (Harbourne 1979; Friend *et al.* 1977; Yates 1982). This may result from direct damage to cellular elements or interference in the normal functions of lung defenses by other agents such as respiratory viruses, and/or indirectly through physiological depression of immunity. Numerous environmental and animal-related factors such as concurrent diseases and stress from severe weather, overcrowding, harassment, heavy intestinal or lungworm infestations and poor nutritional condition appear to have significant effects on the incidence of pasteurellosis in both domestic ruminants and bighorn sheep (Thomson *et al.* 1975; Blood *et al.* 1983; Spraker *et al.* 1984).

Once the bacteria can multiply in an individual, transmission of large numbers of organisms to other individuals occurs by oral or nasal transfer of saliva or nasal secretions. Conditions allowing close contact within the herd increases the possibility of transmission (Yates 1982; Dungworth 1985).

Systemic disease, or what is referred to as haemorrhagic septicemia (Potts 1937; Thorne *et al.* 1982), may also occur in bighorn sheep. Although this condition and its clinical signs are well recognized in domestic animals, documentation of its occurrence in free-ranging wildlife is poor, probably because of its rapid clinical progression and mortality. Pneumonic pasteurellosis in bighorn sheep can have a similar peracute clinical course, even in well observed captive animals. In most instances,

affected wild sheep are found dead, preventing the observation of the progression of clinical signs.

Pasteurella haemolytica pneumonia does produce recognizable pathologic lesions in most animal species. These are characterized by acute fibrinonecrotizing to subacute fibrinopurulent pneumonia and fibrinous pleuritis. Chronic lesions may progress to abscessation and the formation of fibrous pleural adhesions. A variety of bacteria with or without *P. haemolytica* may be cultured from pneumonic lungs at this stage.

Actinomyces pyogenes is the agent recovered next most commonly from pneumonia in bighorn sheep. *Pasteurella* spp. and *A. pyogenes* are frequently found in combination in subacute and chronic bronchopneumonias of domestic sheep and cattle (Dungworth 1985). *Actinomyces pyogenes* causes sporadic infections in most animal species. It is considered to be a normal superficial inhabitant that can cause suppurative lesions of the skin and lungs as well as other tissues if permitted entry. Infections are often localized by the formation of abscesses. Mortality associated with the organism is usually due to chronic debilitating infections. The classic pulmonary lesions associated with the organism are marked suppurative inflammation of bronchioles or the formation of pulmonary abscesses.

Paratuberculosis or Johne's disease is a chronic, progressive condition caused by *Mycobacterium paratuberculosis*. The organism is ingested from a contaminated environment and multiplies within the intestinal mucosa and associated lymphoid tissues, causing focal to diffuse granulomatous inflammation. The infection may resolve or develop over months or years into an invariably fatal clinical course once intestinal absorption is severely affected. Animals become markedly emaciated and often have watery diarrhea before death.

Domestic and native wild ruminants are susceptible to the disease. It is considered to be widespread in some populations of cattle, sheep and goats; however, the inaccuracy of antemortem diagnostic tests has prevented gathering accurate prevalence data (Blood *et al.* 1983). Spontaneous cases of Johne's disease are reported in mountain goat (*Oreamnos americanus*), llama (*Lama glama*), moose, white-tailed deer and elk (Williams *et al.* 1983). It has never been diagnosed in Canadian bighorn herds but has been found repeatedly in two free-ranging bighorn herds in Colorado (Williams *et al.* 1979).

Williams *et al.* (1983) successfully transmitted a bighorn isolate of *M. paratuberculosis* to bighorn-mouflon hybrids, mule deer and elk. The latter species, as well as those previously mentioned, can potentially share ranges or contact bighorn sheep at low or high altitudes. Severe lesions considered capable of shedding large numbers of organisms were present in a mountain goat from a herd in contact with the infected bighorn herds (Williams *et al.* 1979), and the possibility of cross transmission was

raised. It is therefore likely that close contact between infected and susceptible wildlife or domestic species could allow transmission of this disease and its maintenance in herds of bighorn sheep.

Glaze *et al.* (1982) reported a high prevalence of dental abnormalities and osteomyelitis of the mandibles of Dall sheep. Similar lesions were reported to be present in Rocky Mountain bighorn populations of Alberta (Green 1949) and Stone's sheep (*Ovis dalli stonei*) in northern British Columbia (Howe 1981). The bony enlargements were referred to as lumpy jaw, a term usually reserved for the lesions caused by *Actinomyces bovis* in cattle. *Actinomyces bovis* however, has never been identified in mandibular lesions in wild sheep. *Actinomyces pyogenes*, *Fusobacterium necrophorum* and miscellaneous bacteria have been cultured from lesions in sheep in Alaska (Neiland 1972) and the Yukon (Glaze *et al.* 1982). The pathogenesis of these lesions may be presumed to be similar to that of actinomycosis in cattle. It is postulated that conditions such as accelerated tooth wear by glacial silt contamination of forage, coarse vegetation and calcium imbalances in the diets of Dall sheep may predispose to mandibular infections (Glaze *et al.* 1982). Transmission of these bacteria between bighorn sheep was thought to occur at mineral licks and waterholes in Banff National Park (Green 1949).

Lesions of the oral cavity may allow the entry of other bacteria such as *Fusibacterium necrophorum*. This organism is a common secondary invader causing a condition recognized as necrotizing stomatitis or necrobacillosis. Necrotic stomatitis may remain superficial, extend into deeper tissues and bone or reach the lower gastrointestinal tract (Barker and Van Dreumel 1985). The condition is recognized widely in cattle, domestic sheep, deer, reindeer (*Rangifer tarandus*) and elk, and has been seen sporadically in other species (Wobeser *et al.* 1975; Rosen 1981).

Necrosis and acute inflammation of the lower limb and hoof are also associated with *F. necrophorum* as one form of foot rot in domestic animals. It appears to be the major cause of foot rot in wild ruminants. Outbreaks of necrobacillosis have been reported when winter conditions force dense feeding situations, particularly in areas where previous domestic animal or wildlife cases were present. Foot rot epizootics have occurred when animals were concentrated in contaminated moist areas after periods of hot weather. Caking of limbs with mud was believed to allow entry of organisms (Rosen 1981). Neither footrot nor necrotizing stomatitis are reported to be major disease problems in wild sheep species, except for the previous references to occasional diagnoses. It is, however, conceivable that given similar predisposing factors, the same potential exists for the development of these changes on a wider scale.

Other agents capable of initiating respiratory disease syndromes in domestic sheep, as well as other species, include *Chlamydia psittaci* and *Mycoplasma* spp. (Page and Erickson 1969; Blood *et al.* 1983).

Chlamydia psittaci has been isolated from the nasal cavity of normal wild sheep (Pearson and England 1979). The organism is known to be involved in pneumonia in domestic sheep and is considered to be ubiquitous with low host specificity. Subclinical chlamydial infections occur in many animal species and the organism may be harboured in the intestinal tract of some ruminant species.

Mycoplasma arginini was consistently cultured during an acute outbreak of respiratory disease in captive Rocky Mountain bighorn sheep (Woolf *et al.* 1970; Woolf and Kradel 1973) in addition to assorted species of opportunistic bacteria. Although mycoplasma are prominently involved in respiratory diseases of other species (Blood *et al.* 1983), they are also isolated from the upper respiratory tracts of clinically normal animals (Springer *et al.* 1982; Yates *et al.* 1983b) without association with pathologic lesions. In the bighorn outbreak it was not possible to determine the specific significance of bacterial and mycoplasma agents; however, it seems clear that mycoplasma must be included as an agent potentially involved in bighorn disease outbreaks.

PARASITES

Bighorn sheep are hosts to a variety of types of parasites including arthropods, protozoa, cestodes and nematodes (Cowan 1951; Buechner 1960; Becklund and Senger 1967; Uhazy *et al.* 1971; Uhazy *et al.* 1973; Kistner *et al.* 1977). Parasitism may produce deleterious effects on host species through several mechanisms. These may vary with the species and virulence of the parasite, but include previous exposure of the host to the parasite, infection of the host by microorganisms carried by the parasite, inordinately heavy infective doses of organisms and the effects of heavy infections on the host during times of specific physiological demands, such as poor nutritional condition, lactation, during breeding season or with concurrent disease.

Disease syndromes resulting from these direct or indirect effects of parasites on the host are reported in many wild species (Sweatman 1971; Dieterich 1981; Thorne *et al.* 1982); however, only a few species have been reported to be serious pathogens in Rocky Mountain bighorn sheep. Those most commonly associated with clinical signs or mortality in bighorns are *Psoroptes* spp., the scabies mites, and *Protostrongylus* spp., nematodes of the respiratory tract (Buechner 1960; Lange *et al.* 1980; Spraker 1979). Sporadic disease and mortality due to other parasites are also reported (Thorne *et al.* 1982). Other more subtle losses in the form of reductions in growth rate and physical condition are well documented in domestic livestock (Soulsby 1982). These effects are suspected but are as yet undocumented in free-ranging wild sheep.

Arthropods

The commonly recognized external parasites of wild sheep include several species of ticks and mites. These arthropods live for all or part of their life cycle on or within the superficial layers of the host's skin surface feeding on blood or skin debris. Hard ticks, ***Dermacentor andersoni*** and ***D. albipictus*** and the soft-bodied spinose ear tick, ***Otobius megnini*** are found on bighorn sheep as well as other big game and livestock species (Becklund and Senger 1967; Thorne ***et al.*** 1982).

Large numbers of blood-feeding hard ticks may weaken a young or debilitated animal through energy losses assocated with irritation and hair loss, as well as by the volume of blood consumed. ***Dermacentor albipictus***, in particular, is characterized by heavy feeding of adults during the winter months when hosts can least afford either blood or hair loss (Drew and Samuel 1985). ***Otobius megnini*** may cause marked irritation and possible hearing impairment in heavy infections when exudate is deposited in the ear canals of infected individuals (Thorne ***et al.*** 1982). ***Dermacentor andersoni*** and ***O. megnini*** can also secrete neurotoxins capable of causing paralysis leading to death of the host (Blood ***et al.*** 1983). Multiple host ticks such as ***D. andersoni*** can serve as vectors for the transfer of infectious diseases, including Rocky Mountain spotted fever, where these diseases are endemic (Thorne ***et al.*** 1982).

All three tick species have been identified throughout bighorn sheep ranges in the U.S.A. and Canada; however, no comprehensive surveys have defined the full extent and effects of tick infections in bighorn sheep. It is believed that ticks alone do not cause major health problems in wild sheep, although their effects on animals suffering from other conditions may be considerable.

Buechner (1960) and others (Becklund and Senger 1967; Lange ***et al.*** 1980; Foreyt ***et al.*** 1985) have identified various mite species of the genus ***Psoroptes*** on Rocky Mountain, Mexican and desert bighorn sheep throughout southwestern U.S.A., in Wyoming, Montana and Idaho. Epizootics of severe skin lesions (scabies) in bighorn sheep caused by these mites have occurred since at least the mid-1800s. In addition, there have been many reports of animals with milder ear, head and neck infections caused by ***Psoroptes***, rather than by ***O. megnini*** ear ticks (Ward 1915; Foreyt ***et al.*** 1985).

The introduction of domestic sheep into bighorn habitat has been linked with the outbreaks of scabies in wild sheep (Buechner 1960). Due to the difficulties in identification of mites, it is not yet clear which species of ***Psoroptes*** mites occur on wild sheep, which have caused outbreaks of scabies and whether they originated from domestic stock.

Psoroptes ovis mites, common to domestic sheep, cattle and horses (Sweatman 1971) and *P. equi* var. *cervinus* are those species implicated in causing clinical scabies and ear infections in bighorn sheep. Mites taken from scabies cases in New Mexico were tentatively identified as *P. ovis* (Lange *et al.* 1980). These mites did not infect domestic sheep, cattle, elk or mule deer (Thorne *et al.* 1982). The mite was suspected to be specific to the bighorn sheep, but further transmission studies are required to prove this point.

In 1978, significant clinical signs of scabies were seen in large numbers of Mexican bighorn sheep (*Ovis canadensis mexicana*) for the first time in New Mexico (Lange *et al.* 1980). This major outbreak was the first to occur in recent times, to be well investigated and the first to be treated. Animals with alopecia and pruritus over a large percentage of the body and crusty, suppurative skin lesions of the ears were seen during the winter months. Sheep in various stages of emaciation were observed by the early spring, and less than half the expected number of sheep were seen in the following year. Lange *et al.* (1980) attributed the reduction of sheep to either mortality, although few carcasses could be located in the rugged terrain, or to mass movement of sheep from traditional range. No direct evidence of dispersion was reported, but this has occurred in other areas in response to changes in feed and water availability (Watts and Schemnitz 1985). Eventually, Lange *et al.* (1980) determined that scabies alone was responsible for mortality of 60-70% of the New Mexico sheep. The outbreak was believed to be controlled by mass capture and medication of a majority of the population (Kinzer *et al.* 1983).

Sweatman (1971) suggested that the effects of mite infections were more severe during the winter months in part as a result of reduced nutritional quality of forage. His suggestion was supported by experiments with domestic sheep in which mange skin lesions caused by *Sarcoptes* spp. mites were less severe in sheep fed diets supplemented with trace mineralized salt and carotene. The lesions became more severe when animals were also infected with high levels of gastrointestinal parasites which were believed to decrease nutrient absorption (Sweatman 1971).

Protozoa

The most significant protozoal parasite of bighorn sheep are coccidia of the genus *Eimeria*. This parasite undergoes a direct life-cycle within the intestinal epithelium of the host animal. Pathogenic effects vary from inapparent to peracute disease, or chronic villous atrophy from continual sequential reinfections (Barker and Van Dreumel 1985). Coccidia are considered host specific with a number of species described for each host species.

Coccidiosis is characterized as a disease of herd animals where most animals are infected but few develop clinical disease. Animals are usually

infected as juveniles and, once recovered from the self limiting course of disease, are rarely seriously affected again by the same species of *Eimeria*. High densities of animals on a small area increase the concentration of infective oocysts on contaminated forage. Under these conditions, clinical disease may become widespread. Outbreaks in cattle and domestic sheep occur in late fall and winter, especially in overcrowded situations with animals stressed by cold or other diseases (Blood *et al.* 1983). High mortality may also occur in animals lacking previous exposure and infected with large numbers of organisms.

There have been a number of changes in the taxonomy of *Eimeria* and confusion in species nomenclature, particularly in the older literature, has resulted. The *Eimeria* species in Rocky Mountain bighorn sheep have been identified as *E. ahsata*, *E. arloingi*, *E. crandallis*, *E. ovinoidalis*, *E. faurei*, *E. granulosa*, *E. intricata* and *E. parva*. These were found in surveys of bighorn sheep in Colorado and Wyoming (Becklund and Senger 1967), and all have been reported in Canada (Uhazy *et al.* 1971). The first four species are considered pathogenic in domestic sheep (Blood *et al.* 1983).

In domestic lambs, typical signs of coccidiosis include weakness, inappetance and weight loss with soft or liquid feces. Bloody diarrhea is present in severe cases, but is less frequent than in bovine coccidiosis (Blood *et al.* 1983). Pout (1976) found that with natural multiple *Eimeria* species infections, the growth rate of lambs was not affected. Further experiments suggested that in lambs, other pathogenic factors such as concurrent enteric infections with bacterial or viral agents must be involved for conversion to clinical disease (Pout 1976).

Coccidiosis due to mixed infections has occurred in captive bighorn sheep ranging in age from 4 months to young adults (Thorne *et al.* 1982). Clinical signs ranged from inapparent infection to passage of soft to liquid feces. Severely ill sheep usually had concurrent respiratory or systemic diseases. The disease in free-ranging bighorn sheep is unlikely to be a serious health problem but may be a secondary disease in weakened, crowded animals.

Sarcocystis spp. are protozoa with an obligatory two-host life-cycle. Sporocysts passed in feces of a definitive carnivorous host contaminate forage or water. Ingested infective forms undergo merogony in endothelial cells and mature as cysts in the skeletal or heart muscle of an intermediate herbivorous host. More than one species commonly infects livestock and wildlife hosts at the same time. There is believed to be a high degree of host specificity (Thorne *et al.* 1982).

Many *Sarcocystis* spp. are considered to be non-pathogenic in the intermediate host (Dubey and Fayer 1983); however, those with canid definitive hosts are more likely to cause clinical disease (Blood *et al.* 1983). Experimental infection of mule deer fawns with high numbers of

sporocysts from a dog host has caused serious illness and death with widespread hemorrhage, anemia, lymphadenopathy and emaciation (Kistner *et al.* 1977).

Increasing levels of *Sarcocystis* infection were present during the fall and winter in naturally infected mule deer in Oregon (Dubey and Kistner 1985). The timing of the most severe infections coincided with the period of most severe weather and, hence, nutritional demands. Kistner *et al.* (1977) suggested that the poor physical condition of some individuals in the spring was due to *Sarcocystis* spp. infections.

A single species of *Sarcocystis*, *Sarcocystis ferovis*, has been definitively identified in Rocky Mountain bighorn sheep (Dubey and Fayer 1983). Muscle cysts were also present in Dall sheep (Dau 1981). Dubey and Fayer (1983) were unable to transmit *S. ferovis* to domestic sheep, goats or cattle, but proved that the coyote (*Canis latrans*) could serve as the definitive host. Domestic lambs may be seriously affected by *S. tenella* (Dubey and Fayer 1983), but nothing is known of the effects of this parasite on bighorn sheep. Muscle cysts are commonly found and, although no studies of the epizootiology or pathogenicity of these parasites in bighorn sheep have been done, they may cause similar effects on heavily infected wild sheep.

Helminths

The final major group of parasites that occur in bighorn sheep is composed of cestodes and nematodes.

Generally, the presence of helminths is far more common than the diseases they cause. The diagnosis of disease induced by these parasites, or helminthiasis, should be reserved for cases where sufficient numbers of parasites are present, pathological lesions that can be attributed to the parasites are present and there are clinical symptoms associated with the pathogenic mechanism of the infection (Barker and Van Dreumel 1985).

The helminths of the gastrointestinal tract have been divided into five categories on the basis of feeding mechanism and pathogenic effects on the host tissues (Barker and Van Dreumel 1985). The first group is composed of those parasites which attach at a single point but are otherwise wholly lumen dwelling. Nutrients are absorbed passively from the host gut content. If the parasite is present in large numbers, subclinical diseases such as poor growth, diarrhea or obstruction may occur rarely. Tapeworms of the small intestine such as *Moniezia* are the best examples of this group.

A second group, composed of nematodes such as *Haemonchus* spp., damages the mucosa in order to consume blood. Clinical signs are associated

with blood loss and hypoproteinemia. Secondary inflammation of the eroded mucosa may also occur.

The third group can be described as affecting the functional integrity of the intestinal mucosa and causing protein loss, malabsorption and diarrhea. This is the result of chronic irritation by parasites within crypts or within the epithelial cell layer. The response consists of the invasion of lymphocytes, plasma cells and eosinophils, goblet cell metaplasia, crypt hyperplasia and villous atrophy. A large number of species, including members of the genera *Ostertagia*, *Marshallagia*, *Trichostrongylus*, *Cooperia*, *Trichuris* and *Nematodirus*, belong to this group.

Nematode species that burrow into or erode the mucosa and submucosa belong to the fourth group. *Oesophagostamum* spp. and others may incite protein losses and inflammatory lesions by this mechanism.

The final group causes lesions in tissues other than the gastrointestinal tract, usually due to normal, or aberrant, larval migration. Larval cestodes or nematode larvae in accidental hosts can cause lesions at various extraintestinal sites.

Representatives of each of the five groups may be present in a host simultaneously. The effects of each parasite species may be impossible to distinguish from one another, particularly if they belong to the same pathogenic group.

The cestodes, or tapeworms of wild ruminants, have relatively complicated live-cycles involving two or more hosts. The definitive hosts of the family *Taeniidae* are wild or domestic carnivores. Larval stages are present in wild or domestic herbivores. Two of the most common species in game animals are *T. hydatigena*, which forms hollow cysts or cysticerci in the liver and abdominal cavity, and *T. krabbei* larvae that infect skeletal or heart muscle (Leiby and Dyer 1971). Degenerating cysticerci are often present as fibrotic nodules within the liver, whereas larger cysts may be attached to mesenteries. Secondary symptoms may result due to the location of cysts, expansive growth in organs or heavy infections.

Bighorn sheep in Colorado, Montana and Wyoming have been found infected with *T. krabbei* (Thorne *et al.* 1982). The cysticercus of *T. hydatigena* or "*Cysticercus tenuicollis*" is the most common larval tapeworm of bighorn sheep. This parasite has been reported to occur in Dall sheep, California, desert and Rocky Mountain bighorn sheep of the U.S.A. (Marsh 1938; Philip 1938; Allen 1955; Kistner *et al.* 1977; Dau and Barrett 1981), British Columbia and Alberta (Cowan 1951; Blood 1963). There is no direct evidence of mortality in bighorn sheep associated with these parasites, but heavy infections inflicting extensive damage to the liver and

peritoneum have been found in bighorn and domestic sheep (Thorne *et al.* 1982; Blood *et al.* 1983).

A second group of cestodes, including ***Moniezia***, ***Thysanosoma*** and ***Wyominia species***, utilize wild and domestic ruminants as definitive hosts, occupying sites in the small intestine and bile ducts. Intermediate hosts are free- living mites in the case of ***Moniezia*** infections. The live cycles of ***Thysanosoma*** and ***Wyominia*** are poorly defined. These tapeworms have been reported to cause diarrhea and weight loss in young pronghorn antelope and mule deer fawns, but, although they have also been found in Rocky Mountain bighorn sheep, no evidence of clinical disease was present (Thorne *et al.* 1982). They have been identified in Montana, Colorado, Wyoming (Becklund and Senger 1967), British Columbia and Alberta (Cowan 1951).

Thysanosoma actinoides causes mild to marked fibrosis, inflammation and bile duct hyperplasia of the common bile duct in bighorn sheep. Large numbers of adult worms may partially occlude the flow of bile, but no clinical signs have been reported, even with heavy infections. ***Wyominia tetoni*** occupies the intrahepatic biliary system as well as the common bile duct, so that pathologic changes are similar, but more extensive than with *T. actinoides*. ***Wyominia tetoni*** is more widely distributed than *T. actinoides*, being reported in Dall sheep, California, desert and Rocky Mountain bighorn sheep in nine states (Becklund and Senger 1967; Colwell *et al.* 1975; Kistner *et al.* 1977; Thorne *et al.* 1982), British Columbia, Alberta and the Yukon (Cowan 1951; Gibbs and Fuller 1959; Blood 1963).

Nematode parasites comprise a large group of tubular organisms that inhabit various body systems of animals. Most nematodes are associated with the gastrointestinal tract and their pathogenic mechanisms have been outlined. Certain species occupying the respiratory system are of particular importance to wild sheep and will be discussed in detail later.

Gastrointestinal nematodes are so common that they are considered ubiquitous. Their effects on the gut and the health of the host are proportional to the number of parasites, their mechanism of feeding and other host-related factors such as animal density, immune and nutritional status, climate and the presence of secondary microorganisms (Soulsby 1982).

Nematode infection of the gastrointestinal tract, or nematodiasis, is a significant clinical and economic problem in domestic livestock. The high prevalence of infection exists primarily because of intensive management practices. Clinical nematodiasis is most frequently seen in young, growing animals, but may occur in any age group where conditions favour the parasite. This same pattern of disease may also be presumed to occur in wildlife species.

A large number of gastrointestinal nematode genera have been identified in California and Rocky Mountain bighorn sheep and include *Ostertagia*, *Marshallagia*, *Haemonchus*, *Skrjabinema*, *Nematodirus*, *Cooperia*, *Capillaria*, *Trichuris*, *Oesophagostamum*, and *Trichostrongylus* (Becklund and Senger 1967; Boddicker and Hugghins 1969; Uhazy *et al.* 1971; Kistner *et al.* 1977; Thorne *et al.* 1982). Becklund and Senger (1967) found that 70% of 51 species of bighorn nematodes were also parasites of domestic sheep and 35% of cattle. It is likely that in areas where livestock and bighorns graze on the same ranges at different times of the year, some species of nematodes are interchanged as animals move through contaminated pastures.

Clinical nematodiasis in bighorn sheep appears to be rare. Consistent pathological changes have been identified for some nematodes and are believed to cause significant effects on specific individuals and groups of bighorn sheep. Part of the third or fourth larval stage of *Ostertagia* and *Marshallagia* spp. is spent within the abomasal mucosa. Nodular inflammatory lesions are produced by the burrowing larvae to give a classic "moroccan leather" appearance as has been described for domestic ruminants (Blood *et al.* 1983). Large numbers of larvae and adults may severely damage the mucosa and glandular elements. The resulting protein loss and diarrhea cause weight loss in domestic or wild ruminants alike (Soulsby 1982; Thorne *et al.* 1982; Blood *et al.* 1983).

The presence of *Haemonchus*, *Skrjabinema*, *Nematodirus*, *Cooperia*, *Capillaria*, *Trichuris* and *Oesophagostamum* spp. may cause microscopic and physiological changes in the intestinal tracts of affected bighorn sheep in a manner similar to that of Trichostrongylid-type outbreaks on domestic sheep (Soulsby 1982). They do not appear to be very significant in most free- ranging bighorn sheep; however, most reports of gastrointestinal nematode infections of bighorn sheep describe the occurrence, rather than the clinical and pathological effects of these parasites.

LUNGWORM

The wild sheep of North America are definitive hosts for several species of lung nematodes. *Protostrongylus stilesi* Dikmans, 1931, and *P. rushi* Dikmans, 1937, were first described by Dikmans in the lungs of Rocky Mountain bighorn sheep from Colorado in 1930, Wyoming in 1934 and in Dall sheep by Goble and Murie in 1942 (Forrester 1971). The host range has since been extended to include California and desert bighorn sheep (Cowan 1951; Blood 1963; Forrester 1971).

Dikmans (1935) found *P. stilesi* adults and larvae in nodules within the lung parenchyma, whereas those of *P. rushi* were larger and grossly visible within bronchioles and bronchi (Dikmans 1943). A third species, *P. frosti* Honess, 1942, also present in parenchymal nodules, was described by Honess (1942) in bighorn sheep from Wyoming. Few specimens were

available, however, and Dougherty and Goble (1946) suggested that this species was indistinguishable from *P. stilesi*. Later reviews concluded that *P. frosti* should be considered a valid species (Forrester 1971). Despite this conclusion, it has never been reported outside of Wyoming. The only other recognized definitive host for protostrongylid lungworms is the mountain goat. Both *P. stilesi* and *P. rushi* have been reported in mountain goats from western Canada (Cowan 1951) and the U.S.A. (Buechner 1960) and lesions of bacterial pneumonia with lungworms, similar to those in bighorn sheep, have been diagnosed occasionally (Joslin 1986).

The second lungworm genus found in bighorn sheep is *Muellerius*. *Muellerius capillaris* Mueller, 1889, is considered to be a mild pathogen of domestic sheep and goats (Dungworth 1985). It is not commonly found in bighorn sheep, but was reported as the only lungworm species present in Rocky Mountain bighorns from South Dakota (Pybus and Shave 1984). Larvae with dorsal spines resembling those of *Muellerius* have been found in Dall sheep (Goble and Murie 1942) and bighorn sheep of British Columbia, Alberta (Hudson *et al.* 1971), Montana and North Dakota (Pybus and Shave 1984), but conclusive species identification has not been made.

Demartini and Davies (1977) suggested that heavy infections of *M. capillaris* played a role similar to that of protostrongylid lungworms and predisposed a group of recently captive bighorns to the development of bacterial pneumonia. No evidence of *P. stilesi* was found in these sheep. *Protostrongylus* spp., however, remain by far the most common and important parasite of bighorn lungs. References to lungworm in the remainder of this paper will therefore concentrate on those species.

Protostrongylid lungworms are, and have probably always been, an integral element of the bighorn sheep respiratory disease complex. Buechner (1960) reviewed the literature on bighorns to that date and postulated that lungworm infections "acted as predisposing agents which condition the lungs for pneumonia". His historical references to lamb and adult bighorn mortality caused by lungworm or verminous pneumonia were extensive. He cited reports of these animals in poor physical condition with chronic pleuropneumonia and varying degrees of lungworm infection. Many early reports did not distinguish deaths as a result of bacterial pneumonia from those complicated by lungworm infections; however, it seems likely that lungworms were at least present in bighorn sheep prior to Dikman's (1935, 1943) description and contributed to pneumonia outbreaks.

Marsh (1938) cultured *Pasteurella* and *Actinomyces* bacteria in association with lungworm infections in bighorn dieoffs in Montana and Wyoming. Reports of subsequent bacterial pneumonia outbreaks in these and other areas consistently identified lungworm infections of various degrees of severity (Buechner 1960). Because of the constant relationship of bacteria

and lungworms, pneumonic conditions of bighorn sheep have been referred to as the lungworm-pneumonia complex (Forrester 1971).

Clinical symptoms and mortality caused by lungworm are far less common than is the presence of the parasites. Lungworm infection, or protostrongylosis, is almost ubiquitous. Herd infection rates of over 90% have been commonly reported in Rocky Mountain and California bighorn sheep throughout western Canada (Blood 1963; Uhazy *et al.* 1973) and northwestern U.S.A. (Forrester and Senger 1964; Kistner *et al.* 1977; Thorne *et al.* 1982). Low and zero infection rates have only been found in herds of desert bighorn sheep from hot, semiarid regions of southwestern U.S.A. (Allen 1955), probably due to a lack of suitable habitat for the intermediate hosts of the parasite in these areas.

The two-host development of *P. stilesi* and *P. rushi* infections is ideally adapted to the maintenance of the parasite in populations of bighorn sheep. The life-cycle of both species is considered similar in respect to all but one phase. The apparent ability of *P. stilesi* to infect bighorn lambs *in utero* provides an extremely important mechanism for establishing early infections in neonatal sheep and initiating fatal respiratory disease in lambs less than 6 months of age. *Protostrongylus rushi* is believed to be less important in the pathogenesis of pneumonic disease since it is not known to infect the fetus and is less invasive of pulmonary tissues.

Adult *P. stilesi* lungworms are present in dorsal caudal lung lobes. Eggs are deposited in adjacent alveoli, develop to first stage larvae and migrate to small air passages. Larvae move into proximal airways, are swallowed and passed in fecal pellets. At this stage the larvae are extemely resistant to environmental conditions (Forrester and Senger 1963). Further development requires larval penetration into one of several species of tiny land snails. Suitable snail intermediate hosts are usually common on bighorn ranges and occupy microniches at the base of forage plants eaten by bighorn sheep.

After penetration into the intermediate host, larvae molt to the third and infective stage and remain viable, presumably for the life of the snail. Infected snails are accidentally consumed with forage, particularly when high density sheep populations graze pastures closely. Larvae penetrate the gut wall and migrate to the lungs for maturation to adults. Within 28 to 35 days after infection, first stage larvae ap ear in the feces (Spraker 1979).

Ingested third stage *P. stilesi* larvae also have the unique capability to lie dormant in the lungs, or possibly other body tissues, of pregnant ewes with preexisting lungworm infections (Hibler *et al.* 1974; Gates and Samuel 1977; Spraker 1979). No further development occurs until the last 6 weeks of gestation. The larvae then enter the maternal circulation and migrate to the uterus where they cross the placenta and enter

the liver of the fetus. At the birth of the lamb, the larvae recommence migration and simultaneously reach the lungs. Adult *P. stilesi* are present in lungs by 20 days after birth, with first stage larvae appearing in the feces by the time the lamb is 6 weeks of age.

The reactions of host tissues to lungworm infections and the clinical syndromes produced vary with the number of lungworms and the stage of the life-cycle that is present. Protostrongylosis of young lambs is the classic example of the pathogenic effects of the parasite. Spraker (1979) followed the development of transplacentally transmitted *P. stilesi* larvae to explain the high mortality of lambs in Colorado herds. He described the mass movement of third stage larvae from the liver into the lungs soon after birth. Multifocal, sometimes massive, pulmonary hemorrhage was caused as large numbers of larvae left the circulation and occupied alveoli. Infective larvae reaching the lung, after ingestion in either lambs or adults, probably enter the lung more sporadically but develop in a similar manner.

The parasites concentrate in the dorsal caudal lung lobes. Grossly visible nodules are raised above the pleural surface. In acute infections the nodules are grey-yellow and translucent. The nodules become progressively more firm, opaque and white, then contract as fibrous tissue proliferates within alveolar walls and the pleura.

The initial cellular reaction to the larvae developing into adults is limited to mononuclear cells. When eggs and first stage larvae develop, intense infiltrations of lymphocytes, macrophages and eosinophils develop. Alveolar walls thicken with the mononuclear cells, increased smooth muscle and fibrous tissue hyperplasia. The overlying pleura and surrounding interlobular septae become increasingly fibrotic. Adjacent alveoli are usually atelectatic. Focal necrosis and acute inflammation with neutrophils may be present when larvae invade airways for movement up the bronchial tree. Bronchiolar smooth muscle is often hyperplastic and the epithelium frequently becomes hyper and/or metaplastic. Peribronchiolar lymphoid tissue is usually prominent within the nodules and may be found at some distance from the nodule. A mucoid exudate, often containing larvae, is usually present in the lumen of airways.

In lambs or adult bighorn sheep, larvae in larger airways can be accidentally aspirated into more cranial, dependent lung lobes. These larvae are rapidly surrounded by inflammatory cells consisting mostly of macrophages and multinucleate giant cells. The granulomas initiated by degenerating larvae are extensive if large numbers of larvae are aspirated. This frequently appears to be the case in neonatal lamb protostrongylosis.

The pathogenesis of protostrongylosis in both lamb and adult bighorn sheep involves mild to extensive damage to the lower respiratory tract. If tissue damage is sufficient and pulmonary defenses are compromised by

lungworms, viral or bacterial infections or other concurrent parasitic, nutritional or physiological stressors, opportunistic bacteria colonize the damaged lungs and cause typical lesions of the respiratory disease complex of bighorn sheep.

Since the health of an individual sheep may be so profoundly affected by the presence of large numbers of lungworms, numerous studies have attempted to assess the lungworm burdens of bighorn sheep. The number of first stage larvae in the feces has been associated with other physiological indices in order to describe the overall parasite and health condition of bighorn herds.

The degree of lungworm infection in an animal is proportional to the availability of infective intermediate hosts, the density of infected animals utilizing the range and the degree of infection in other animals in the herd, in addition to other host factors as previously discussed. Fecal larval output undergoes seasonal and annual fluctuations. The time of maximum counts varied between herds in several studies (Forrester and Senger 1964; Uhazy *et al.* 1973; Holmes and Samuel 1974). The highest mean number of lungworm larvae per gram of feces was usually seen during the late spring or in winter. The pattern of larval output variations has also changed in subsequent years in the same herds (Gates 1975).

Much of the variation in fecal larval output appears to be associated with changes in the availability of the intermediate host and the state of development of the larvae within the snail. Snails with third stage larvae must occupy sites used by sheep for ingestion and infection to occur. Snails prefer the high moisture and moderate temperatures that also favour the movement of larvae from fecal pellets, thereby increasing the snail infection rate (Forrester and Littell 1976). Snail numbers and activity were higher during spring months with high rainfall, and sheep fecal larval output was high in the following fall, than in years with lower spring rainfall (Lange 1973; Gates 1975; Forrester and Littell 1976). Appropriate conditions are well provided in areas of sheep habitat that are preferentially used. These areas, such as those near bedding sites, invariably green up early in the spring, are heavily grazed and are believed to be foci for transfer of infected intermediate hosts (Lange 1973). Samson and Holmes (1985) found that most first to third stage development of larval *Protostrongylus* spp. occurred during the warm summer months. Almost no development occurred during the winter. Infected snails containing third stage larvae on ranges during autumn and early winter are then probably responsible for a large proportion of the transmitted lungworm.

Forrester and Senger (1964) advised the consideration of larvae count fluctuations when assessing the lungworm infection levels of individuals or herds. They felt that the examination of a small number of fecal samples provided the closest estimation of infection rates. Uhazy *et al.* (1973)

and Holmes and Samuel (1974) suggested that the proportion of heavily infected animals in a herd contributed another useful index of population condition. It appears that larval production may be governed by changes associated with the individual herds' environmental conditions, host nutrition, pregnancy or other physiological demands, as well as by the biology of the lungworm. Use of several indices to lungworm numbers to estimate the levels in sheep herds must then be carried out over several years to be of value.

MISCELLANEOUS

Several other disease conditions are reported and may contribute to sporadic mortality in bighorn sheep. None of the conditions appear to have a specific infectious etiology.

Amyloidosis is a systemic disease characterized by the extracellular deposition of amyloid, an eosinophilic, homogenous, fibrillar protein. The cause of excess protein production is usually idiopathic; however, it may be produced by abnormal plasma cells or, in the secondary form, as a byproduct of chronic inflammation (Maxie 1985). The most common sites of deposition are within renal glomeruli or visceral organs such as the spleen and liver. Severe cases in aged domestic animals may cause proteinuria and terminal renal failure.

Amyloidosis has been infrequently reported in free-ranging wild animals. It was seen in three emaciated, wild, adult Rocky Mountain bighorn sheep from Montana and Idaho (Hadlow and Jellison 1962). Two of these sheep had chronic frontal sinusitis and the third chronic bacterial leptomeningitis. Seven of 17 bighorn sheep with chronic bacterial pneumonia from a dieoff of captive sheep had similar lesions (Woolf and Kradel 1973). The condition was also reported in desert bighorn sheep (Cosgrove and Satterfield 1982). All lesions occurred mainly in the kidney, liver, spleen and adrenal glands, and were consistent with secondary amyloidosis. Hadlow and Jellison (1962) remarked on the apparently high incidence of cases in wild sheep as compared to other game species, and speculated that bighorn sheep may be prone to develop amyloidosis.

Inflammation of the nasal passages and sinuses has been reported in desert bighorn sheep (Cosgrove and Satterfield 1982) and bighorn sheep in Wyoming and Montana (Honess 1942). The condition is similar to sinusitis in domestic sheep and is associated with secondary bacterial invasion of tissues damaged by larvae of the nasal bot fly *Oestrus ovis*. Sinusitis appears to be a sporadic disease in wild sheep.

Capture myopathy is a degenerative disorder of cardiac or skeletal muscle seen commonly in exertionally stressed or recently captured wildlife. The affected animal may suffer from a variety of signs, including those related to primary muscle lesions, and to the secondary sequellae of shock or

kidney failure. The condition is associated with prolonged muscle exertion, fear, handling and transportation, high environmental temperatures and low vitamin E-selenium levels (Spraker 1976; Chalmers and Barrett 1982). Myopathy may also predispose free-ranging animals to predation, since similar muscle changes have been present in animals after pursuit by predators or hunters (Harthoorn 1975; Chalmers and Barrett 1982).

Three clinical and pathological syndromes of capture myopathy were seen in adult bighorn sheep captured and transported for 18-24 hours (Spraker 1976; Demartini and Davies 1977). Some sheep died within 12 hours with few gross lesions; others were ataxic and had myoglobinuria before death within 24 hours. Myonecrosis and nephrosis were the major lesions in these animals. The third group of sheep had bilateral ruptures of the gastrocnemius muscles due to widespread myonecrosis 48 hours after capture. Capture myopathy in wild sheep appears to be significant primarily during live capturing procedures. Wild and domestic carnivore or human-related harassment may also initiate the condition, but is probably sporadic in free-ranging animals.

The diagnosis of neoplasms in wildlife is relatively uncommon. Benign and malignant tumors are sporadic and may or may not contribute to mortality. The effects of each tumor is proportional to the extent and organ(s) of involvement. Several types have been reported to occur in wild sheep. Dieterich (1981) found tumors of an "uncertain histologic type" in a Dall sheep in Alaska. An intestinal adenocarcinoma was present in a bighorn X mouflon hybrid, and pituitary and adrenal adenomas were found in Rocky Mountain bighorn sheep (Thorne *et al.* 1982). None of these appeared to have any significance other than to the affected individual.

RESPIRATORY DISEASE IN DOMESTIC ANIMALS

CURRENT CONCEPTS - AGENT SYNERGISM

Jakab (1982) and Yates (1982) reviewed the clinical and experimental literature on viral-bacterial interactions in respiratory diseases of domestic and laboratory animals. It has been demonstrated in many species that some virus-induced respiratory infections have an overwhelming suppressive influence on pulmonary defense systems and allow colonization of certain bacteria and the development of pneumonia.

In the normal lung, the mucociliary apparatus, alveolar macrophages and circulating leucocytes combine to intercept, inactivate and eliminate aspirated bacteria or foreign particles. Local and systemic immunoglobulins assist in local defense and particle ingestion. The pathogenesis of acute viral respiratory infections involves the modification of these reactions. This may include necrosis of epithelial cells of all levels of airways, enhanced susceptibility to bacterial attachment and colonization and numerous dysfunctions of alveolar macrophages (Green *et al.* 1977; Warr and Jakab 1979; Yates 1982). Pulmonary responses produce an inflammatory cell invasion of peribronchiolar tissues and airway lumina, and filling of alveoli with edema fluid and leucocytes, secondarily impairing antibacterial functions at this level.

The secondary infection of lung tissues by bacteria occurs only under certain conditions. Respiratory tract defenses must be affected at all levels to allow pathogenic bacteria to multiply within the upper respiratory tract, reach, invade and multiply in distal air passages and alveoli. It appears from experimental studies, that only with properly timed, large doses of virus do the pulmonary antibacterial defenses fail completely to allow the rapid growth of bacteria and the production of fatal pneumonia.

In the natural state, however, other factors must be considered to act as concomitant stressors with viral infection to reduce the efficacy of antibacterial defenses of the lung. Rarely do pure infections occur as in experimental situations. There is much evidence that other agents, such as mycoplasmas and chlamydia, other concurrent diseases and environmental stressors assist in decreasing resistance to secondary bacterial invasion.

Combined infections of viral and bacterial agents in domestic sheep and cattle are associated with initiating more severe respiratory disease than the disease caused by either pathogen alone (Friend *et al.* 1977; Warr and Jakab 1979; Jericho *et al.* 1982; Yates 1982; Trigo *et al.* 1984). Initially, studies of the most widely researched livestock respiratory disease complex, bovine shipping fever, concentrated on reproduction of the disease using sequential infections of PI3 virus and *P. haemolytica*

(Lopez *et al.* 1976; Friend *et al.* 1977). Infectious bovine rhinotracheitis (Jericho *et al.* 1976; Jericho and Langford 1978; Yates *et al.* 1983a), RSV (El Azhary *et al.* 1981), BVD, adeno-, rhino- and reoviruses (Yates 1982) are now all known to be involved in bovine respiratory diseases and may play roles in the development of shipping fever.

Synergistic effects of PI3 or RSV and *P. haemolytica* have been demonstrated and were highly reproducible in both immunocompromised and conventionally reared domestic lambs (Sharp *et al.* 1978; Rushton *et al.* 1979; Davies *et al.* 1981; Trigo *et al.* 1984). Pneumonia of varying severity proportional to the experimental conditions were present, i.e. more severe in immunocompromised animals. Experimental infection with an ovine isolate of RSV alone, and with *P. haemolytica*, produced only mild clinical respiratory symptoms in 3 month old domestic lambs (Trigo *et al.* 1984). However, lesions were more severe and there were increased numbers of inflammatory cells in the dual infection. Although the results did not indicate that this virus was a significant pathogen under the experimental conditions, Evermann *et al.* (1985) suggested that with intensification of management, stressful conditions or multiple bacterial or viral infections, the susceptibility of sheep to RSV may increase. These authors also speculated that, because this virus was antigenically related to bovine RSV, there was definite potential for interspecific transmission.

Much time and money have been spent to elucidate the synergism of viral and bacterial agents in respiratory diseases. Despite the advances in identification of organisms and definition of immunological dysfunctions, the mechanisms linking the interactions of host, pathogen and environment to cause disease are still not completely understood in domestic species. Although extrapolations to wild species may be made and may be applicable, experimental studies are obviously needed before the mechanisms may be understood in wild sheep.

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WR-23 Critical habitat of caribou (*Rangifer tarandus caribou*) in the mountains of southern British Columbia. K. Simpson, K. Hebert, and G.P. Woods. February 1987. 13pp.

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WR-24 Impacts of a hydro-electric reservoir on populations of caribou and grizzly bear in southern British Columbia. K. Simpson. February 1987. 40pp.

WR-25 The effects of snowmobiling on winter range use by mountain caribou. K. Simpson. February 1987. 15pp.

WR-26 Quesnel Highlands wolf control project. D. Hebert. January 1987. 10pp.

WR-27 Muskwa Wolf Management Project of northeastern B.C. 1986-87 annual report. J.P. Elliott. April 1987. 20pp.

WR-28 Vancouver Island wolf control project. Year 1 progress report. D. Janz. July 1987.

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